considered the possible catastrophic sequelae of further episodes of autonomic dysreflexia, our patient decided to abstain from further sexual activity.

REFERENCES

1 Mathias CJ, Frankel HL. Autonomic disturbances in spinal cord lesions. In: Mathias CJ, Bannister R, eds. *Autonomic Failure: A Textbook of Clinical Disorders of the Autonomic Nervous System*. Oxford: Oxford University Press, 1999:494–513

2 Kewalramani LS. Autonomic dysreflexia in traumatic myelopathy. *Am J Phys Med* 1980;**59**:1–21

3 Lindan R, Joiner E, Freehafer AA, Hazel C. Incidence and clinical features of autonomic dysreflexia in patients with spinal cord injury. *Paraplegia* 1980;**18**:285–92

4 Kurnick N. Autonomic hyperreflexia and its control in patients with spinal cord lesions. *Ann Intern Med* 1956;**44**:678–85

5 Steinberger RE, Ohl DA, Bennett CJ, McCabe M, Wang SL. Nifedipine pretreatment for autonomic dysreflexia during electroejaculation. *J Urol* 1990;**36**:228–31

6 Frankel HL, Mathias CJ. Severe hypertension in patients with high spinal cord lesions undergoing electroejaculation: management with prostaglandin E2. *Paraplegia* 1980;**18**:293–9

7 Hussain IF, Brady CM, Swinn MJ, Mathias CJ, Fowler CJ. Treatment of erectile dysfunction with sildenafil citrate (Viagra) in parkinsonism due to Parkinson's disease or multiple system atrophy with observations on orthostatic hypotension. *J Neurol Neurosurg Psychiatry* 2001;**71**:371–4

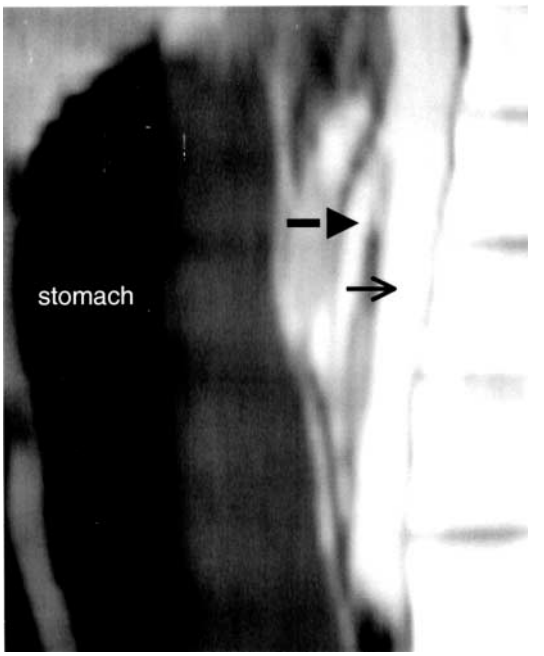

Figure 1 **Sagittal CT scan showing narrow angle between aorta (light arrow) and superior mesenteric artery (heavy arrow)**

Superior mesenteric artery syndrome in a patient with HIV

T Agarwal MS MRCS T A Rockall MD FRCS
A R Wright MRCP FRCR[1] S W T Gould BSc FRCS

J R Soc Med 2003;**96**:350–351

One of the contributing factors in superior mesenteric artery syndrome is severe wasting.

CASE HISTORY

A man of 27 with established AIDS was referred with a diagnosis of cerebral toxoplasmosis and clinical features of high gastrointestinal obstruction. He had been vomiting profusely for six weeks and was unable to retain anything orally. He now weighed just 30 kg. Inability to retain his antiviral medication had led to worsening immunosuppression, and severe oral and oesophageal candidiasis prevented him swallowing his own saliva. The cerebral toxoplasmosis had resulted in mild left sided hemiparesis. On examination of the abdomen there was epigastric fullness with a positive succussion splash.

Ultrasound showed the stomach to be massively distended, and CT demonstrated obstruction of the third part of the duodenum by extrinsic compression. A narrow aorto-mesenteric angle in the sagittal section was highly suggestive of superior mesenteric artery (SMA) syndrome (Figure 1) and hypotonic duodenography showed typical cut-off at the third part of the duodenum.

An attempt was made at conservative management, initially with total parenteral nutrition and later with nasogastric feeding. However, weight loss continued and the nasogastric aspirate remained greater than 1L/day. Enteral access was necessary for administration of his antiviral and antitoxoplasmosis medication. Surgical intervention was therefore required. The options included a feeding jejunostomy alone (to improve the patient's nutritional and immune status) or a definitive procedure for SMA syndrome. The latter was chosen. Intraoperative findings confirmed the presence of SMA syndrome. The stomach and proximal duodenum were distended and there was visible compression of the third part of the duodenum,

Academic Surgical Unit and [1]Department of Radiology, St Mary's Hospital, London W2 1NY, UK

Correspondence to: Mr Tushar Agarwal, Academic Surgical Unit, 10th Floor QEQM Wing, St Mary's Hospital, Praed Street, London W2 1NY, UK

E-mail: atushar13@hotmail.com

the site of block being confirmed by nasogastric air insufflation. Because of anatomical and technical considerations, a gastrojejunostomy was performed instead of the more commonly advocated duodenojejunostomy. Postoperatively, the nasogastric aspirate decreased rapidly and from day 2 the patient started tolerating oral fluids. His weight increased and his viral load decreased when antiviral medication was restarted. The toxoplasmosis and oesophageal candidiasis resolved.

COMMENT

SMA syndrome (also known as Wilkie's syndrome[1]) has many causes including high insertion of the duodenum at the ligament of Treitz, a congenitally low origin of the superior mesenteric artery and compression of the duodenum due to peritoneal adhesions as a result of duodenal malrotation.[2,3] However, the most common cause is thought to be narrowing of the aorto-mesenteric angle causing extrinsic compression of the third part of the duodenum.[4]

Predisposing factors include severe wasting diseases such as burns or malignancy, severe trauma (especially head injuries), spinal trauma (cast syndrome), eating disorders and the postoperative state. The condition is difficult to diagnose as it mimics gastric outlet obstruction. Hypotonic duodenography and CT scan can be helpful.

Treatment includes an attempt at feeding to improve nutrition. If this fails, various surgical procedures have been advocated. Duodenojejunostomy is the definitive procedure of choice. However, other procedures including gastrojejunostomy, Roux-en-Y duodenojejunostomy and anterior transposition of the third part of the duodenum have been reported.

In the present patient, the development of SMA syndrome exacerbated the wasting of AIDS by preventing oral antiretroviral therapy. When conservative measures proved unsuccessful, decisions on surgical management had to take account of the risks to operating personnel. Standard precautions in high risk patients include the use of impermeable gowns, double gloving and eye protection and immediate disposal of sharps in a sharps bin. Only the necessary number of assistants, who should have an appropriate level of experience, are allowed to scrub for the surgical procedure. Surgical staplers are used in preference to sutures. In the present case the gastrojejunostomy could have been done laparoscopically. This would have greatly lessened the exposure risk from sharps injury, though a theoretical risk is aerosolization of infected material through the laparoscopic ports during instrument changes. We decided against a laparoscopic procedure because we had planned to do a duodenojejunostomy (a technically demanding procedure when done laparoscopically). If we had originally intended to do a gastrojejunostomy, laparoscopic surgery would probably have been our choice.

REFERENCES

1 Wilkie DP. Chronic duodenal ileus. *Am J Med Sci* 1927;**173**:643–9

2 Cohen LB, Field SP, Sachar DB. The superior mesenteric artery syndrome: the disease that isn't or is it? *J Clin Gastroenterol* 1985;**7**:113–16

3 Strong EK. Mechanics of arteriomesenteric duodenal obstruction and direct surgical attack upon etiology. *Ann Surg* 1958;**148**:725–30

4 Ylinen P, Kinnunen J, Hockerstedt K. Superior mesenteric artery syndrome—a follow up study of 16 operated patients. *J Clin Gastroenterol* 1989;**11**:386–91

A diabetic patient with recurrent tetraparesis

Yusuf A Rajabally MD

J R Soc Med 2003;**96**:351–352

The most frequently encountered neuropathy in diabetes is slowly progressive. Sensory symptoms predominate, sometimes accompanied by autonomic involvement, and motor deficit is usually absent.

CASE HISTORY

A man of 65 was referred after rapid onset of weakness in the arms and legs. It was his second such episode. 8 years previously, when he was 57, non-insulin-dependent diabetes mellitus had been diagnosed, and a year after onset he had developed a mild axonal diabetic polyneuropathy, causing distal paraesthesiae. Insulin was required from 2 years after diagnosis. He was hypertensive, treated with a combination of a diuretic, a calcium channel blocker and an alpha adrenoreceptor blocker. At age 60 he experienced weakness affecting all four limbs, slowly progressing over four months. There was no respiratory or bulbar involvement. He reported a concomitant increase in his sensory disturbance—ascending paraesthesiae in the four extremities. The symptoms had appeared despite adequate glycaemic control with insulin and gliclazide. On

Department of Neurology, University Hospitals of Leicester, Royal Infirmary, Leicester LE1 5WW, UK

E-mail: yusuf.rajabally@uhl-tr.nhs.uk